The Nature of Entropy in Frozen Fluids Provides Clues to Key Aspect of Aging Process

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Introduction

In the cryo-preservation of human tissues, most tissues will not stand up to freezing due to the expansion of water when transitioning into the frozen state. This author recently published a concept that would enable the use of Coulomb Force Lines to raise the freezing point of water artificially to above the temperature of the human body, rendering the process safe (as the fluid would contract rather than expand if forced into the frozen state at a higher temperature.)

Abstract

Certain types of cells can survive traditional cryo-preservation, such as sperm cells and even fertilized embryos. The presence of large quantities of proteins which act a bit like anti-freeze in these special cells mean that preservation of these particular cells by freezing is possible. In these cases, interestingly, unusually low temperatures are required. Even when these cells are in a frozen state, their entropy does not entirely cease. Sperm cells, for instance, must be preserved at -80°F or below for long-term viability. While this fact about protecting reproductive cells is well-known, no one seems to be asking why it is the case that harmful entropy occurs at temperatures higher than -80°F. In solving the mystery as to why this is so, I have answered a question about entropy generally, and the aging process, more specifically.

If entropy is not entirely halted by the state or condition of fluids being locked into a lattice structure i.e. being in the frozen state, in what dimension can a molecule move when its position relative to other molecules is fixed? The answer is that molecules such as water molecules may gradually move about, provided the presence of some degree of phononic energy, by rotating on their own axis within the lattice.

Occasionally, a water molecule in such a lattice may, rather than being pinned to the lattice by a Coulomb line that runs through the exact center of the molecule i.e. the oxygen component, a water molecule may be offset with one of its hydrogens being the portion pinned to the lattice by the force lines. Add to this the possibility of the molecule twisting on its own axis gradually and it becomes possible for a hydrogen to be kicked off of the water molecule upon thawing. Ordinarily, hydrogens in water tend to be liganded at an orientation of 104.45° of angle relative to their associated oxygens. As previously hypothesized, grabbing these hydrogens with Coulomb lines and abruptly changing their orientation relative to one another would have the potential to kick a hydrogen off of the molecule, meaning that such a technique (when dealing with liquid) could be used to efficiently harvest hydrogen.

If this can be true for liquid water in an artificial hydrolysis mechanism, it

stands to reason that this sort of dehydrogenation process could be transpiring on a sporadic basis naturally in both frozen and liquid biological tissues and could underpin a key aspect of the aging process.

In lipid structures such as cellular and nuclear membranes, chains of proteins are interlinked ultimately by covalent electron bonds between water and other molecules comprising the proteins. Since 2017, it has been understood that senescent cells feature a wrinkling of the nuclear membrane, but it is not known to the scientific community what causes this wrinkling.

If these covalent bonds are based upon; at the most fundamental level; the repulsion of electrons and other electrons, then the forces at work are fundamentally the same as in the case of whole molecules locked into a lattice in the case of frozen water. This would mean that rotation on the molecules' own axis is essentially the only respect in which such molecules are free to move with relation to the entirety of the protein structure.

In such a structure, even if it is not frozen, phononic activity may, on rare occasion, cause a single water molecule within a protein link in a nuclear membrane to rotate sharply enough that a hydrogen is kicked off, rendering the molecule as a hydrogen monoxide.

A hydrogen monoxide can do the job of maintaining the integrity of the membrane i.e. it can keep foreign materials out of the protected nucleus, however, the crucial function of allowing duplicated DNA to egress from the nucleus as part of the cell replication process cannot proceed when the water molecules that make up the lipid membrane lack even a single hydrogen at the chosen point of permeation. If DNA attempts to pass through such a membrane at a point in that proverbial chain link fence where a link is missing a hydrogen, the fence will not perform its task of allowing the transport of the DNA, slowing the healing process and further damaging the membrane in one fell swoop. Once damaged in this manner, the membrane appears under a microscope to be wrinkled and the cell is therefore considered senescent.

Senescent cells are capable of spawning new cells, but these cells are generally also senescent and this process is generally slower than healthy cell replication since attempts to pass DNA through corrupted sections of nuclear membrane necessitate that part of the genetic code of the new cell be re-encoded and re-transmitted repeatedly until a healthy section of membrane is selected by chance and the process may be completed in its entirety. Since senescent cells must copy their own DNA to facilitate replication about 5x as often as healthy cells, in this author's view, the paradox of senescent cells' higher risk of carcinogenesis despite a slower apparent rate of replication can be explained by this higher level of activity in the nucleus.

Recent experimental therapies have purportedly been effective in reversing cell senescence. I would propose that the as-yet unknown mechanism underpinning these treatments must fundamentally work by introducing free hydrogen ions to the nuclear membrane with those hydrogens linking up with hydrogen monoxides and restoring them to their original status as standard water. These therapies, although promising, are incomplete, both because the wrinkling of the nuclear membranes would not be entirely "ironed out" by

rehydrogenization alone and because aging is a multifaceted process. The other major component of aging is systemic waste protein buildup secondary to the kidneys reaching their fundamental limit for storage of these proteins, as described in a previous publication by this author.

This hypothesis is consistent with what is already understood with regard to psychological and physiological stress as it relates to aging. It is already widely accepted that cell senescence, including that associated with premature graying of hair, is induced by stress; the exact mechanism however, is still a matter of some debate.

Stress leads to the release of hormones such as adrenaline, which signal to cellular mitochondria to become hyperactive. The mitochondria, which are located within the cell, under normal conditions, produce only modest amounts of heat in short bursts. Under stress conditions, these heat-generating organelles produce much greater amounts of heat than normal.

This increased heat generation creates a sharper thermal gradient within the cell itself, meaning that the nuclear membrane is unevenly heated under this condition. This gradient can give rise to torsion of the lipid proteins, leading to a higher chance of a dehydrogenation event within the water molecules in the nuclear membrane's lipid proteins.

Phonons are the only force occurring naturally in this context capable of applying sufficient torsion to cause the release of hydrogen from water through the forced proximal approach of the two hydrogens of water to one another.

Conclusion

By filling in a missing piece of the puzzle concerning how even a frozen biological tissue can experience corruptive entropy, we gain a key insight into a major component of aging.